

ORGANOCHLORINE RESIDUES IN BLOOD PLASMA OF MIGRATING PEREGRINE FALCONS

Charles J. Henny

U.S. Fish & Wildlife Service, Patuxent Wildlife Research Center,
Corvallis, Oregon

F. Prescott Ward

Toxicology Branch, Chemical Systems Laboratory, Aberdeen Proving
Ground, Maryland

Kenton E. Riddle

University of Texas Cancer Center, Veterinary Resource Division,
Bastrop, Texas

Richard M. Prouty

U.S. Fish & Wildlife Service, Patuxent Wildlife Research Center,
Laurel, Maryland

Abstract: Peregrine falcons (*Falco peregrinus*) were captured and bled during fall migration at Assateague Island, Maryland/Virginia, and at Padre Island, Texas, from 1976 through 1979. In addition, spring migrants were captured and bled at Padre Island, Texas, from 1978 through 1980. More than 400 blood plasma samples were collected and analyzed for organochlorine pesticides and polychlorinated biphenyls (PCB's). Blood plasma residue comparisons are made between fall migrants of the various sex and age classes captured along the Atlantic Coast and the Texas Gulf Coast. Also, residue comparisons are made of fall migrants versus spring (after a winter south of the U.S. border) migrants in Texas. Some recaptured individuals that we had previously bled provided additional information for interpreting residue patterns over time. Finally, for the purpose of residue interpretation, an adjustment equation is used to project residues from the blood plasma of adult females to estimated residues in eggs laid by the peregrines. Previously banded peregrines captured and bled during this study included birds from Alaska, Yukon, northern Alberta, Mexico, and Cornell-released birds from the East Coast.

FATAL AVIAN POX IN BALD EAGLES FROM ALASKA

Sheila K. Schmeling

U.S. Fish and Wildlife Service, National Wildlife Health
Laboratory, Madison, Wisconsin

Douglas Docherty

U.S. Fish and Wildlife Service, National Wildlife Health
Laboratory, Madison, Wisconsin

Abstract: Within the last 2 years, three immature bald eagles (*Haliaeetus leucocephalus*) have been found alive in Alaska with massive cauliflowerlike head lesions. These proliferative lesions were large enough to cause visual impairment in all of the birds. The three were emaciated and died within a few days of capture. Gross and histologic findings were typical of pox infection. This is the first record of pox infection in the bald eagle.

Avian pox, characterized by discrete, proliferative lesions on the skin or the mucous membranes, has been reported in several raptorial species. This disease is caused by a member of the genus avipoxvirus (Matthews 1979).

Pox has been observed in captive raptors such as the red-tailed hawk (*Buteo jamaicensis*) by Halliwell (1972), peregrine falcon (*Falco peregrinus*) by Cooper (1969), and Saker falcon (*Falco cherrug*) by Greenwood and Blakemore (1973). Moffatt (1972) has reported avian pox in a free-flying juvenile golden eagle (*Aquila chrysaetos*), and Pearson et al. (1975) published a report of a fatal pox infection in a free-flying rough-legged hawk (*Buteo lagopus*). The following report is the first description of avian pox in free-flying bald eagles (*Haliaeetus leucocephalus*).

MATERIALS AND METHODS

All three of the eagles were kept frozen until their submission to the National Wildlife Health Laboratory (NWHL).

Necropsy

The eagles were thawed and subjected to a routine necropsy. Samples from the lesions were taken for bacterial, viral, and histopathological examination. The brains, livers, and carcass remains were submitted to the Patuxent Wildlife Research Center (PWRC) for routine toxicological analysis.

Histopathology

The tissues were fixed in 10 percent formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin for conventional light microscopy.

Virology

A suspension was prepared from the pox lesions of one eagle and passed through a 0.45 μ filter prior to initial inoculation on the chorio-allantoic membrane (CAM) of 10-day-old embryonated mallard and chicken eggs. One passage of the CAM's harvested from the initial inoculation was made, and these membranes were harvested on day five postinoculation, fixed in formalin, and sectioned for histopathological examination.

Microbiology

Five percent sheep blood agar plates were inoculated with the pox lesion and incubated for 24 hours at 37.5°C. Bacterial colonies were identified on differential media.

RESULTS

Case Histories and Necropsy Findings

All three immature eagles discussed below were extremely emaciated, averaging 3,500 grams, while eagles from Alaska in good body condition averaged 5,000 grams. Pronounced muscular atrophy and fat resorption were evident. The livers and gastrointestinal tracts were atrophied, but there was no evidence of inflammation or necrosis. The lungs and air sacs were normal. Figure 1 shows locations where the three infected eagles were found.

Eagle A

Eagle A was found on 6 November 1978 on Starrigavan Road near Sitka (Figure 1), laying face down, with its head tucked under its body, unable to walk or fly. Massive cauliflowerlike lesions, grayish-white in color, and 1 to 1.5 cm in height, covered the head (Figure 2). The left eye was totally covered by a 8 x 12 cm lesion, while the eye was

only partially occluded. There were lesions along the side of the mouth but none were visible in the oral cavity. A 2 cm diameter lesion was present on the ventral surface of the neck. One foot had a 0.5 cm raised ulcerated area. Most of the numerous lice on the head and neck were identified as Craspedorrhynchus haliyeti, with a few Degeeriella discocephalus discocephalus. This immature male was euthanized.

Eagle B

Eagle B died during the night of 10 January 1979 after being found in a weakened state earlier that day near Thorne Bay, Prince of Wales Island. This immature male's head lesions were smaller and more discrete than those found on A or C. On the right side of the bill was a growth 2.5 cm in diameter and 1 cm high, extending 1 cm into the oral cavity. A 1 cm diameter lesion was noted under the mandible, while the tongue had a 0.5 cm diameter lesion on both the top and bottom surfaces. There were seven raised areas, measuring from 0.5 to 1.5 cm in diameter, impairing vision in the right eye.

Eagle C

Eagle C, an immature female, was found on 8 August 1980 near Anan Creek. The large head lesions were diagnosed as avian pox, with secondary bacterial infection. She was given penicillin and lincocin, sprayed for lice, and had maggots removed from tracts in the head lesions. The bird ate, but became steadily weaker and died on 10 August.

The most prominent of the cauliflowerlike head lesions were a 5 x 8 x 2 cm lesion which obscured most of the right eye, a large coalescing lesion, 8 x 10 x 2 cm, which covered the left eye and extended onto the bill, and a 4 cm pedunculated lesion under the mandible. There were smaller lesions on the roof of the mouth and on the tongue.

Laboratory Findings

Histopathology

Histopathological examination of fixed sections of the lesions from all three eagles showed hyperplastic squamous epithelial cells with eosinophilic, intracytoplasmic inclusion bodies typical of avian pox (Karstad 1971). Scattered areas of necrosis and bacterial infiltration were present.

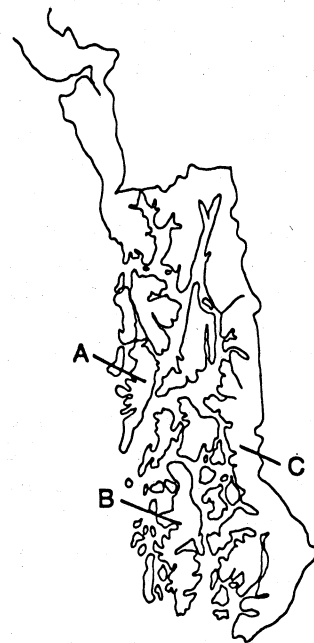


Figure 1. Southeastern Alaska. Collection location of eagles A, B, and C with avian pox.



Figure 2. Eagle A. Head pox lesions.

Virology

Gross examination of the chicken CAM's inoculated with material from eagle C revealed generalized thickening near the site of inoculation, suggesting that pox lesions were diffuse.

Histological examination of fixed sections from this thickened area revealed that there was a proliferation of epithelial cells resulting in a thickness of 8-20 cells rather than the usual 2. Individual cells had a large cytoplasmic vacuole which pushed the nucleus to the margin. An eosinophilic proteinaceous body, best described as roughly globular and granular (morulalike) was observed in the cytoplasm of an occasional epithelial cell. Similar inclusion bodies were not typical of pox, suggesting that their formation was in an early stage of development and therefore, incomplete. Supporting connective tissue was heavily infiltrated with heterophils, many of which were necrotic.

No abnormalities were observed on gross or histological examination of the mallard CAM's.

Bacteriology

The dermal lesions from eagles A and B produced mixed bacterial growth. A coagulase-positive Staphylococcus aureus was the only potential pathogen isolated. The intestinal contents were negative for Salmonella sp. Bacterial culture was not attempted on tissues from eagle C because of its prior antibiotic treatment.

Toxicology

Organochlorine analysis at PWRP revealed nontoxic levels of organochlorine pesticide residue in tissues submitted from eagles A and B. Eagle C had higher residues, but the levels were well below those known to cause mortality.

DISCUSSION

The gross and histopathologic findings in these three eagles were typical of avian pox (Karstad 1971). In all cases, the head lesions were extensive enough to have interfered with the birds' ability to locate and capture prey. As a consequence, all three of the birds were emaciated.

There appeared to be secondary bacterial invasion of the pox lesions. Staphylococcus aureus was isolated from two of the birds; the

third had been treated with antibiotics. However, there was no evidence of generalized bacterial infection.

All three of the eagles examined were immature. In chickens (Karstad 1971), mourning doves (Locke 1961), mockingbirds (Kale and Jennings 1966), and albatross (Friend unpublished), avian pox is reported to occur most often in young birds. None of the 872 eagles submitted to the NWHL from the lower 48 states have had pox lesions. Of the 87 eagles submitted from Alaska, only these three had pox lesions, and all three were from the Southeastern Panhandle. In the golden eagle, the only report of avian pox was from a bird in the Prince George area of British Columbia (Moffatt 1972).

Our observations indicate that there is a strain of virulent pox virus that affects eagles. Transmission of pox virus requires either a break in the skin and contact with infected individuals or a mechanical vector, such as the mosquito (Karstad 1971). Those requirements would be met by behavioral and environmental factors and would not be expected to be limited by geographic boundaries. At this time we have no explanation for the very limited distribution of the cases of avian pox described in this report.

ACKNOWLEDGMENTS

The authors wish to thank Dr. Louis Locke for his interpretation of the histology slides and Renee Romaine for her technical assistance in isolating the virus.

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SESSION 6. Techniques for Raptor Management and Study

CHAIRMAN:

RON GARRETT
U. S. Fish and Wildlife Service
Alaska Regional Office
Anchorage, Alaska

